CONNEMARA PUBLIC LIBRARY
7 - DEC 1958
MADRAS

(For the use only of Registered Medical Practitioner or Hospital or Laboratory)

THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH INDIAN MEDICAL ASSOCIATION
(With which is incorporated the "Miscellany")

Vol. XXV NOVEMBER 1958 No. 5

ENTERO-QUINOL

IODOCHLOROXYQUINOLINE TABLETS

For

Amoebic Dysentery and other Intestinal Disorders

★

For further particulars, please write to : -

East India Pharmaceutical Works Ltd.,

CALCUTTA-28.

CONTENTS

1. Review of Dermatology (Contd.)
   by Dr. T. V. Venkatesan ... 129

2. The Problem of Peptic Ulcer
   by Dr. K. A. Kalyanam ... 138

3. Post Traumatic Knee Joint Diseases
   by Prof. Dr. Sophus Vom Rosen ... 145

4. Case Notes ... 150

ELIXIR HEPOSIM WITH EXTRA FOLIC ACID & VITAMIN B12.

A proteolysed, liver yeast stomach concentrate with the vitamins of the B complex inclusive of FOLIC ACID & VITAMIN B12 for oral administration in gastro-intestinal disorders like sprue-like syndrome and coeliac diseases in children, manifestations of deficiencies of the components of the B complex and also of considerable value in the treatment of general conditions of blood impoverishment to supply the vital substances which stimulate the process of repairs and replacements of the tissues and assist in the maintenance of normal body functions.

Manufacturers :—

THE SOUTH INDIAN MANUFACTURING CO.,
MADURAI.

7 - DEC 1958

(FOR CIRCULATION AMONGST MEMBERS ONLY)

THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH OF THE INDIAN MEDICAL ASSOCIATION

(With which is incorporated the "Miscellany")

Vol. XXV] NOVEMBER 1958 [No. 5

REVIEW OF DERMATOLOGY (*Continued*)

by

Dr. T. V. VENKATESAN, M.B., B.S., F.C.C.P., F.D.S. (London) F.A.I.M.

Ringworm of the Feet and Hands.

The different manifestations may be divided into the following 3 groups.

1. Acute vesiculobullous lesions.
2. Chronic intertriginous lesions.
3. Chronic hyper Keratotic lesions.

1. **Acute Vesiculobullous Lesions:** The usual causative agents are:

(*a*) Trichophyton mentagrophytes and trichophyton rubrum.

(*b*) Epidermophyton floccusum is an occasional causative agent. The essential lesion is a vesicle which may be solitary or multiple. The vesicle is usually deeply situated. The contents are clear unless contaminated. Sensitiveness, pruritis and pain may accompany the lesion.

The various measures are:

1. Warm 1 in 8000 acriflavine or permanganate bath and opening vesicles.

2. The application of hydrocortisone lotion (EFCOKLIN lotion)

3. The systemic administration of corticosteroids.

1. **Chronic Intertriginous Type of Disease:** is seen as maceration and desquamation with or without fissuring between 4th and 5th toes. Frequently small vesicles may be seen at the border. This type of disease is usually associated with secondary infection and lymphangitis. The most effective measures of treatment are:

(*a*) Dryness: These includes most careful and meticulous attention.

(*b*) The application of warm 1 in 8000 permanganate bath.

(*c*) The topical applications are Efcorlin lotion and Tineafax dusting powder.

3. **Subacate or chronic papulosquamous type** of tinea of the hands and feet. This is produced by Trichophyton rubrum. This may also produce Hyperkeratotic lesions. The measures are 6% salicylic acid ointment.

Ringworm of the glabrous skin : (Tinea Caporis or Tinea Cicinota) Clinical variants of Tinea Corporis are :

(1) **Ammular lesions** : This is so called classical ring worm. There is a ring in the centre of which appears clear and the surrounding border is inflammed. The usual causes are Trichophyton mentagrophytes and mocrosperson audouin.

(2) **The Eczematous type** : The lesions are round but scaliness, redness and slight infiltration are present through the lesions. The treatment is similar to ordinary annular lesion.

(3) **Crusted type** : The response to topical treatment is slow at first in this type.

(4) **Herpetic form type** is due to a zoophilic variety. A mound of vesicle and formation of new vesicles are the characteristic features.

(5) **Plaque type** is produced by Trichophyton rubrum. The plaque resemble seborrholic dermatitis with minimal scaling.

(6) **Kerion type of Tinea profunda.** The lesion is ordinarily produced by a zoophilic type of fungus especially Trichophyton verrucosum. The lesion is instensely inflammed. It is elevated circumscribed tumour. At times the entire mass becomes boggy and neurotic. The measures are systemic antimicrobical therapy.

(7) **Majocchi's granuloma** : This is a granulomatous follicullitis due to Trichophyton rubrum. Antifungal agents are of no avail ; but antibacterial and certicosteroid treatment is beneficial.

Tinea Cruris. The condition is more commonly due to epidermophyton floccosum but trichophyton also may be responsible. Several factors favour relapse.

(*a*) Environmental factors as high temperature and sweating.

(*b*) The wearing of athelitic suporters or shorts.

(*c*) Obesity.

Treatment of Tinea Cruris. The patient should have two sets of underwares and use them alternately. The soiled ones are to be sterilised in boiling water. The underware should be loose and absorbent. Nylan drawers are unsatisfactory.

Application :

During day — Undecylinic dusting powder (Tineafax dusting powder)

During night : 6% Salicylic acid, 3% Benzoic acid in toilet lanoline.

If there is no inflammation unguentum cryserobin can be used for shorter periods.

Infection of Nails. The fungus which most commonly infects the nail is the Trichophyton rubrum. The next is the tricophyton menta graphytes. The ringworm of the nail is one of the worst conditions to treat and if a single nail is involved the best procedure is surgical avalsion of the nail. If many nails are involved the following measures are adviced :

1. R/-Lithium bromide 75 gm.
   Water to make 100 cc.
2. Castellani's paint.
3. Brilliant green 1%.
   Hydragyrum perchloride ½%.
   Spirit Oz. 1
   Mix and make into solution.

Pityriasis Versicolor (Tinea Versicolor) : The disease is caused by Microsporon further. The lesions are fawn coloured, palepink or cafeaulait tint, well defined patches. The chest, back and submaxillary area are involved. The factors maintaining this are hyperhidrosis, infrequent washing with soap and water and wearing woollen vests.

Treatment advised : Apply 3% tartaric acid in water ; scrub with rubber brush. After ½ hour apply 20% sodium thiosulphate and scrub with rubber brush.

Erythrasma : Produces brown patches in the genitocrural and auxillary flexures. The lesions are produced by microsperson micutissimmum. The treatment is similar to Tinea Versicolor.

Tinea Flava : This is caused by Melasfurfur. (Microsporon tropica) The disease takes several forms. The commonest type is the appearance of patches of yellowish brown colour. The face the neck, the arms and the trunk are commonly involved.

Treatment :

R/-	Acid salicylic	Gr. 20
	Hyd. perchlor	Gr. 2
	Rectified spirit	os. 2
	Aqua destilled	os. 6 (six)
Mix.		

Tinea Imbriceita : OR TAKELAU RINGWORM) The fungus trichophyton concentricum is found in the tropics. The lesions are patches or concentric scaly rings.

The best application is that of salicylic 15 grains to one ounce of Linamentum sodi.

Trichosporosis : Synonym : Piedra.

The small black nodules occuring on the shaft of the hair is produced by the fungus piedrar hortai. The nodules contain tighter packed dark brown hyphae. The affected part should be bathed twice daily in a lotion containing formalion 1 dr. to 6 ounces of rectified spirit. The condition occurs in South America.

Systemic Fungal Infections.

1. Moniliasis.
2. Coccodomycosis.
3. Blastomycosis, North American, South American.
4. Histaplasmosis.
5. Actinomycosis.
6. Sporotrichosis.
7. Cryptococcosis.
8. Chloro blastomycosis.
9. Mycetoma.
10. Rhino sporidiosis.

Moniliasis. The involvement takes place of the skin, nail, mucous membranes and also systemic. The cutaneous menliasis. Three varities can be described :

(*a*) Monilial intertrigo.

(*b*) Monilial paronychia.

(*c*) Moniliasis of the glabrous skin.

(*a*) Moniliasis intertrigo produces weeping eroded lesions with scallopped border having a collar of overhanging scales and an intensity red base. The commonest site are axillace, grains, inflammonary areas and natal clefts.

(*b*) Monilial nail infection : Moniliai paronychia produces secondary changes in the nail causing ridges, furrows and discoloration.

(*c*) Monilialis of the mucous membranes.

1. **Thrush**: The hall mark of the disease is the development of whitish patches on the mucous membrane of the tongue, mouth and pharynx. Cracks and fissures at the corners of the mouth produces perleche.

2. Moniliasis vaginites and vulvovaginitis: The features are creamy white discharge per vaginum, intense pruritis and the findings of curdy white flakes attached to vagina.

Treatment of moniliasis: The predisposing factors of moniliasis as malnutrition, excessive exposure to moisture and excessive use of antibiotics are to be corrected.

The other measures are: Local application of 0·5 gentian violet, Nystatin or mycostatin ointment.

The administration of mycostatin or Nystatin tablets (500,000 units each tablet) thrice daily is desirable. Two new antifungus agents have been tried in moniliasis. Dose 12 capsules per day each capsule containing 90 mgm of methyl paraben and 22·5 mgm. of propylparaben.

Coccidomycosis: This produces lesions in the skin, lungs, bones and brain. In the skin it produces granulo matous lesions, multiple nodules and in primary pulmonary coccidomycosis Crythemo nodosum like lesions. The treatment is unsatisfactory.

North American Blastemycosis: is caused by blastomyces dermatitidis. This produces pulmonary and cutaneous involvement.

Stilbamidine is the drug of choice. Dose: 1st day 50 mgm. I. V., 2nd day 100 mgm i. v. 3 rd day 150 mgm I. V. continue 150 mgm until a dosage of 3 gm is reached.

South American Blastomycosis: produces uncleative and granulomatous lesions and dissemination by haematoganous route producing lymph glandinvolvement and pulmonary involvement resembling tuberculosis.

Histaplasmosis: produces beningm pulmonary involvement and disseminated pulmonary involvement. The primary lesions of the skin are extremely uncommon.

There is no satisfactory treatment.

Actinomycosis: involves (a) Cervicofacial (b) Thoracic and (c) Abdominal regions cervicofacial involvement produces a lumpy jaw. The thoracic involvement produces parenchymalous involvement of the lungs with secondary infection resembling tuberculosis.

Abdominal actinomycosis involves ileococccal area and sinuses in the abdominal wall.

The other areas involved are the brain and vertebrae Diagnosis is made by the presence of sulphur granules.

Treatment: Isoniazid 10 mgm per kilo of body weight per day for six weeks. Ivocardiasis produces similar involvement but brain involvement is more common.

Sporotrichosis: produces involvement of the skin and lung.

The cutaneous type of lesions are:

1. Lymphatic type.
2. The fixed type producing acne form lesions and Verrucose lesi ons.
3. Haematogenous type producing subcutaneous nodules.

The systemic involvement of the lungs, bone and testes rarely takes place.

Treatment : Iodides.

Chloroblastomycosis : produces mossy, foot due to phialophara verracosa. The foot and ankles are involved producing Verrucose lesions. The best treatment is cautery.

Mycetoma : or Madura foot The hall mark of the disease is nodules and draining sinuses. No chemotherapeutic agents are available.

DERMATITIS AND ECZEMA

50% of all skin cases constitute this group. The word Eczema has become a word for ignorance. It is a word of the man in the street, of the advertiser and of the charlatan. The proper word is dermatitis. There are two factors in the causation of dermatitis namely predisposing and exciting. The morphological changes in the dermatitis are clear cut.

1. Erythema and swelling of the skin.
2. Oozing and/or versiculation.
3. Crusting and scaling.
4. Thickening and evidences and repeated excoriation.
5. Hyperpigmentation scratch papule formation and/or Lichenification.

Causes And Varieties of Eczema : Contact Dermatitis is of the allergic type. At this state a few points on the phenomena of the hypersensitivity reactions are worthwhile. Allergy is a hypersensitivity reaction. It is acquired. This develops in response to a particular substance or one chemically related to it. An allergic phenomenon is an antigen antibody reaction. It is an immunologic phenomenon.

Serum sickness is an allergic response occurring 8 to 10 days after a single dose of serum.

Anaphylactic shock produces vascular collapse, asthma, urticaria, fainting, lessened coagulability of blood coma and death. The foreign protein injected produces sensitisation. This is called sensitising dose or anaphylactogen. This takes about 10 days. Hence another dose in ten days does not produce harmful effects. After 10 days, the incubation period a second dose produce anaphylactic shock. This is called assaulting dose.

Atopic Allergy : To same foreign proteins our body is immune. To others our body resents their presence. This is idiosynceasy or atopy. It is a fierce and frightened attempt on the part of the cells to preserve their chemicalidentity. Atopy is a natural hypersensitiveness in cartrast to anaphylaxis which is induced. A variety of substances produce this :

(a) Food idiosyncrasies,

(b) Drug idiosyncrasies,

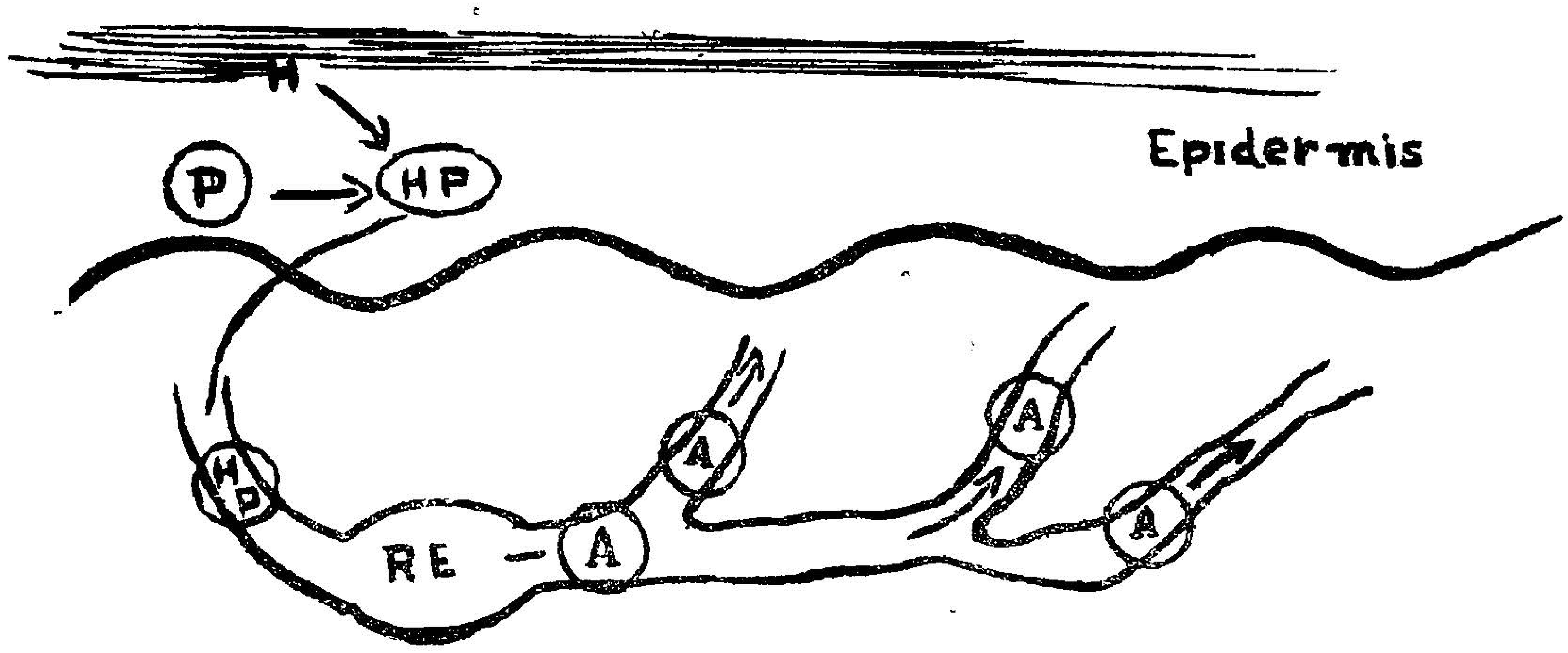
(c) Pollen,

(d) Cutaneous debris of animals etc.

Bacterial allergy is allergy to infection. The tuberculin test is based on this principle. Similarly allergic reactions may occur with fungi, viruses, protozoa and parasites.

Allergic Contact Sensatation : A simple chemical or haptene H applied to the surface combines with an epidermal protein P and form a conjugate HP. This is carried by the lymphatics to the reticuloendothelial cells where special antibodies

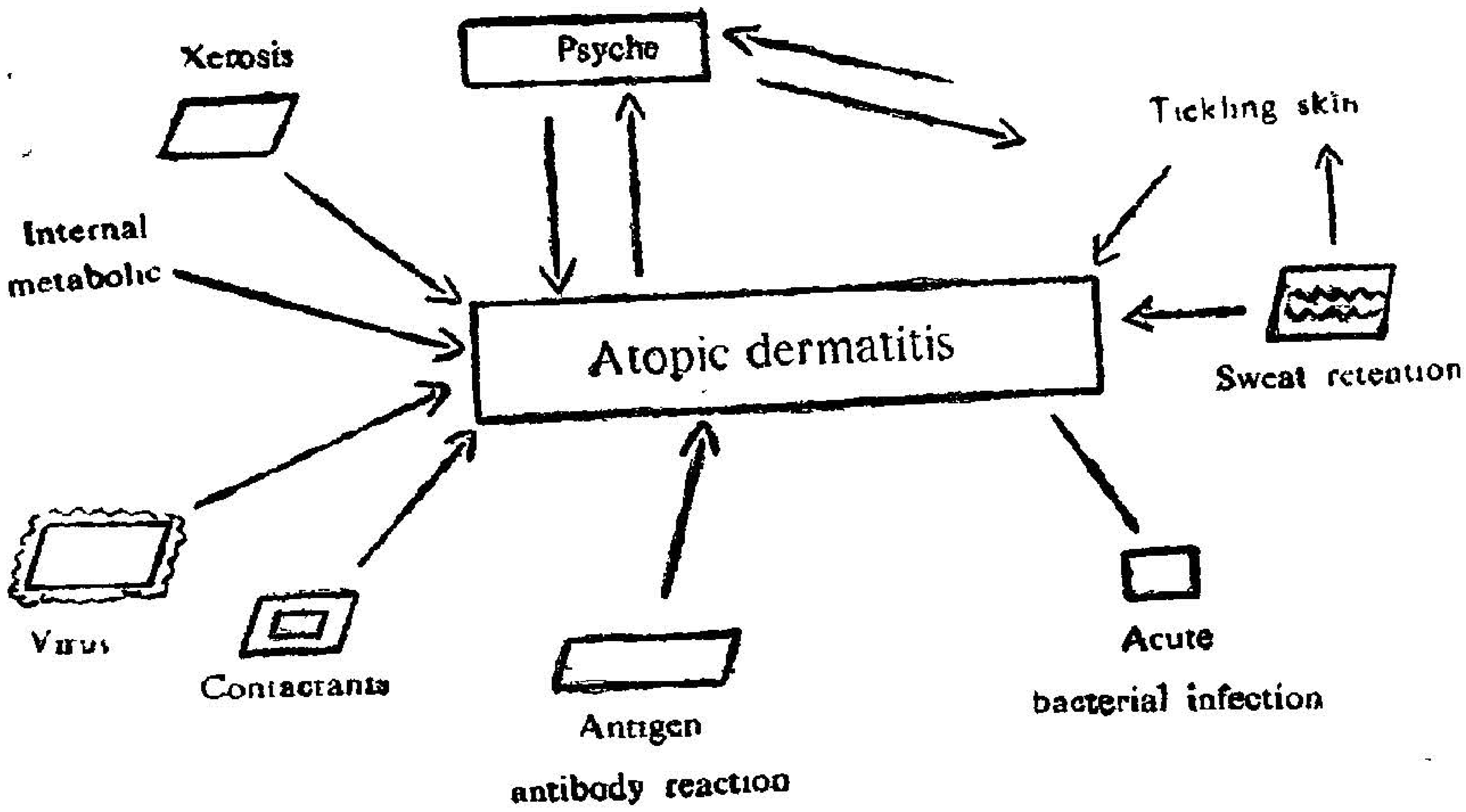
A (Globulins) are formed. These are carried by blood stream to various parts of body and keep the body in a state of cutaneous sensitisation, When haptene comes into contact with the skin allergic contact dermatitis is produced.



Diagnostic features in contact dermatitis: There is fairly sharp delimitation between inflammed and normal skin. The hairy portion of the scalp is rarely involved in contact dermatitis.

Atopic Dermatitis : This is a characteristic recurrent form of eczema' dermatitis.

The following is a principal etiologic and contributory factors :



The Characteristic features of this disease are :

1. Commonest sites are face, near anticubital fossae, hands and wrists, popliteal fossae.
2. There is a family history of allergy as hay fever, asthma, urticaria.
3. Atopic dermatitis is the commonest type of persistance type of infantile eczema.

4. The shock site is the dermis.

5. Pruritis intense and triggered is a common feature.

6. There is marked sweat retention in patients with atopic dermatitis.

7. There are susceptible to disseminated infection as virus of Herpes simplex or vaccinia.

8. A peculiar psychosis is an important feature. An initial marked affability hides a repressed hostility. They tend to be competetive and combative in sexual, Scholastic, recreational and occupational spheres.

9. The physical pattern is peculiar. A dry skin is seen. A characteristic fold of the lower eyelid develop in chronic cases.

Seborrhoeic Dermatitis: The skin is oily with frequent vulnerability to secondary infection by bacterial or candida. There is a Seborrhoeic diathesis.

The factors contributing to exacerbation and persistance of Seborroeic dermatitis are :

1. Acute and chronic bacterial infection.

2. Sweat retention.

3. Mycotic infection and indiscriminate antibiotic therapy.

4. Certain internal diseases are associated with Seborrhoeic dermatitis namely :

   (*a*) Parkinson's disease.

   (*b*) Obese diabetics.

   (*c*) Corticosteroid therapy.

5. Skin diseases associated with Seborrhoeic dermatitis.

   (*a*) Acne vulgaris.
   (*b*) Rosacea.
   (*c*) Psoriasis.

6. The excessive ingestion of oily food stuffy.

7. Seborrhoeic dysfunction.

The sites commonly affected are :

1. Scalp,
2. Forehead above eyebrows,
3. Nasal folds,
4. Retroauricular fold.
5. The intertriginous folds of the trunk namely axillae, under breasts umbilicus, crural folds and gluteal clefts.

Nummular Dermatitis is a distinct member of the eczema dermatitis group producing coin sized lesions. The basic cause of Nummular dermatitis is not known. There is a history of recent contact dermatitis in some cases. There is a reaction to materials absorbed from the primary eczematous patch, Nummular dermatitis is seen more frequently in the aged than in young patients. Bacterial infection and Xerisis predispose to the condition. Psychosomatic factors play an important role. Nummular Dermatitis may appeal suddenly with a shower of lesions.

Lichen Simplex Chronicus or Circumscribed Neurodermatitis has a circumscribed scaling patch with repeated scratching and rubbing. The margins are not fairly sharp. Erythmea and vesiculation are not seen commonly.

Psyche

↓

Lichen chronicus Simplex

↑ ↑ ↑ ↑

Trauma | Nervous flammeus | Chronic bacterial infection | Contactants

Localised neurodermatitis may be initiated or may co-exist with other dermatoses.

Stasis Dermatitis following peripheral venous disease forms a clear cut condition. It is usual by common in the lower limbs. The usual site is above the internal malleolus.

Infantile Eczema is a perplexing and exasperating condition. It commonly occurs in the cheek chin and forehead sparing central area of the face. Secondary bacterial infection may take place. The most common sensitising agent is the cow's milk in these infants. Breast fed infants can also develop a-topic dermatitis.

The other type of dermatitis occurring in children are :

1. Seborrhoeic dermatitis producing cradle cap. In this it is oily scalp with milia and comedenes on the face.

2. An extensive expliative type of Seborrhoeic dermatitis may cccur in infants called Leiner's disease seborrhoeic eczema with involvement of the intertriginas folds is another variety.

Another type of dermatitis occurring in children is called Diaper dermatitis due to diapers.

Therapy in Dermatitis Eczema :

Treatment in acute contact dermatitis The aims are:

1. To provide protection to the damaged tissues.
2. To prevent the excessive accumulation of debris from oozing, scaling and crusting without disturbing normal tissue.
3. To relieve itching and to prevent excoriation.

The Principles of local therapy in all types of dermatitis are as follows :

1. During acute stage without exudation: Zinc cream or calamine cream or antihistamine cream is the best.
2. During the acute stage with excudation calamine lotion is the best.
3. When there is much crusting and oozing use calamine oily linament
4. During the subacute use stage Lassar's paste.
5. During the chronic or Licheified stage use antiparitic ointment with Keralolytics.
6. When there is crusting always remove crusts with starch boric poultice or 2% soda bicarb or 2% sodium sulphate and apply antibiotics for not more than 7 days to prevent sensitivity.

Systemic Therapy : A course of prednisolone orally with three tablet a day for a few days and gradual tailing off dosage is good.

Aetiolotic Therapy : Removal of contactants.

Atopic Dermatitis : The local therapy is similar to systemic therapy. Search for the cause and treat the cause is an easy maxim but difficult to practice. Wherever possible it should be treated.

Non specific desensitisation measures are :

1. Alkalies orally.
2. Antihistamines.
3. Calcium gluconate intravenous with autohoaemotherapy.
4. Peptone injections.
5. Antiallergic treatment with cortico steroids.

Seborrhoeic Dermatitis : Seborrhoea Capits : Shampoo scalp with selenim sulphide and apply externally a lotion containing sulphur, salicylic acid and mercury perchloride.

R/-	Acid salicylic	gr. 20
	Hyd. perchlor	gr. ½
	Oleum Lavender	M. 5
	Spirit Methy	oz. 1
	Oleum Recin	M. 5

The lotion may be made into brillian-tine with Oleum recin m XXX. When the seborrhoeic scaling is thick use the following :

R_I-	Oil of cade	20%
	Sulphur	10%
	Salicylic acid	5%
	Aquaphor	Qs.

Prednisolone and hydrocortisone locally are useful in supressing inflammation.

General measures are :

(1) An Open air life.

(2) Plenty of soap and water, to remove grease.

(3) Diet to contain less cabo hydrate and plenty of fruits and green vegetables.

(4) Ultraviolet radiation.

Nummular Eczema. Local Therapy : During the infected stage a lotion containing neomycin and Hydrocortisone is useful. During the other phase treatment is similar to other type of exzema.

The general measures are

1. Antibiotic therapy.
2. Cortieosterial therapy.

Lichen Simplex Chronicns. The measures are

1. Explanations to the patient that he must not scratch the area lest the skin changes will appear.
2. Local Therapy : Antibiotic lotion and hydrocortisone lotion are useful.
3. Tranquillisers are helpful in this condition.

As a matter of fact tranquilliser have a pride of place in atopic dermatitis too.

Stasis Dermatitis. The measures are suppertive measures with elastoplast and treatment of varicose veins. Unless there is active ulceration Unna's zinc gelatin boot is helpful.

Infantile Eczema : The measures are :

1. To avoid soap. Use starch bath ; soap by its degression and kerato-lytic effect favour the percutaneous penetration of environmental allergens.
2. External application : During acute exudative stage calamine lotion. During acute stage without exudation zinc cream.

   During subacute stage : Antihista-mine cream and Laessor's paste.

   During the Chronic phase : Unguentum crude coal tar or unguentum resorcin may be used.
3. Diet. To avoid cow's milk and use goat's milk or Eledon Aller-gilac.
4. Prednisone and prednisolone are helpful to tide over acute phase.

(Contd.)

THE PROBLEM OF PEPTIC ULCER *

by

Dr. K. A. KALYANAM. F.R.C.S., D.M.R., *Madurai.*

Introduction. This problem though old and ancient is still eluding solution. The rational treatment of any disease depends on a knowledge of its cause and we still know next to nothing of the cause of peptic ulceration. Inspite of this, I feel that we have not conscientiously applied the known facts properly in the management of these unfortunate patients both in the hospital and private practice. All the best people are supposed to have peptic ulcer and these sufferers are on the whole the ablest, the most hard working and the most conscientious members of the community in their period of active life. Their lives are made miserable and their working efficiency is diminished by pain and indigestion.

About 10 per cent of the adult population of the United Kingdom, Continent and States are said to suffer from peptic ulcer, and I am sure, the incidence will almost be the same in this part of our country, though we have no reliable statistics. There is no operating day in any of our hospitals when we do not have a single case of peptic ulcer for operative treatment. So obviously it is a common disease, and it is painful, incapacitating, not free from danger. While not one of the main killing diseases, it ranks high on the list of chronic disabling affections and its frequent occurrence in young adults adds to its social and economical importance. Its distribution is global and no particular race or occupation is exempt, but the commoner incidence in the South and rarity in the North India defies correct explanation.

As regards aetiology, despite much research our knowledge of its causes are still incomplete. But we know the two broad factors viz.

A. The mucosal resistance factor and

B. The acid erosion factor.

It is the product of an abnormal physiology, characterised by inability of a localised area of stomach and duodenum to withstand the digestive action of acid gastric juice. It is true, that the peptic ulcers are found only on surfaces exposed to the actions of gastric juice containing free hydrochloric acid. In ordinary circumstances they are limited to the district between the cardia and bile poppilla: but they may be found in any organ or structure which is brought into contact with gastric juice by sphincteric deficiency. ectopic secretion or surgical anastomosis viz. in the oesophagus, in Meckel's diverticulum or as a stomal ulcer in jejunum or colon. "No acid, no ulcer", is a proved fact as there are no recorded cases of proved ulcer in complete histamine fast achlorhydria. But still acid is the cause of persistance rather that the cause for origin of peptic ulcer. The role of hydrochloric acid in the pathogenesis of peptic ulcer may thus be compared to that of the essential catalyst in a chemical reaction - other aetiological factors (ingredients) are involved in the process, but in the absence of hydrochloric acid the reaction cannot proceed. We know that in gastric ulcer, the output of hydrochloric acid is normal or low and obviously the diminished mucosal resis-

---

* Lecture delivered at Erode Branch of I. M. A, on 6th September 1958.

tance plays the part here. In duodenal ulcer, secretion of Hcl. acid is excessive - 2 or 4 times greater than normal. This hypersecretion is demonstrable for 1 or 24 hours, between meals, during the night and after the ulcer is healed. Its mechanism is not established completely, Anatomically it seems to be correlated with an increased number of parietal cells in the fundus and body of the stomach. It is quantitative and not qualitative as the acid secreted is of same concentration by the individual cell. This total secreted cell mass is actually calculated in the recent work of Professor Card of Edinburgh and the results of operation predicted (common gastroenterology unit in Western General Hospital - Edinburgh). This artificial separation of medicine and surgery is nowhere nor out of place than in gastro-enterology physiologically histamine, acetylcholine, harmonal factors including gastrin from the antrum and adrenocortical hyper function have received consideration. There is no recorded case of peptic ulcer in Addison's disease. Present evidence tends to emphasize the role of vagal hyper activity, acting upon a more responsive gastric secretary mechanism. However other factors chemical and humoral undoubtedly are involved. Though the acid erosion factor may be the predominent cause in Western countries, I feel the mucosal resistance factor plays a more important part in our patients. The resistance of the gastric mucosa to its own secretions, a juice containing powerful enzymes, and inorganic acid in concentration, sufficient to tarnish metals., rot fabrics, is a sufficiently remarkable phenomenon which has excited scientific interests for nearly 200 years. The ulcer is always next to the acid secreting area and never actually involves the acid secreting cells themselves.

Apart from the mucous barrier, scaring and trauma, nutritional disturbances most probably play an important part. Deficiency of Vitamin-C is known to impair wound union by delaying the maturation of collagen while hypo protein aemia and other dietetic defects are responsible for decrease in the mucosal resistance. Even in Western countries though there may not be a primary deficiency. there are untold possibilities of secondary deficiency caused by harmful constituents introduced by modern methods of preserving, preparing and " Improving " - food stuffs.

The aetiology of gastric and duodenal ulcers most probably differ. The gastric ulcer is usually associated with a long stomach of poor tone emptying slowly and a level of acid secretion below the average. while duodenal ulcer is found with a high stomach emptying rapidly with a high acid curve. Duodenal ulcer is definitely associated with a certain physical and psychological type, the lean, wiry, ambitious, worrying, conscientious, heavy smoking intellectual and I do not for one believe that our peasants have no worry, ambition, or psychological or emotional stresses. They have their own problems, worries, and frustrations. Duodenal ulcers seem to be manifestations of the stress syndrome or to use a more modern term, the adaptation syndrome. Though this may not be a predominent factor in our patients, it requires consideration.

On the other surfaces, such as the skin or mucous lining of the mouth, we do not search for one cause of all lesions. Spots on the skin may be due to chemical irritants, thermal or mechanical injury, drugs, local or general infections, or specific fevers. The spots heal and leave no trace behind them ; but if they occurred on the surface exposed to digestion they would persist and assume a uniform pattern.

" An automobile may break down for many reasons. A tyre may go flat, a wheel may come off a bolt may drop from the steering, a fly may choke a jet,

or a drop of water may block a petrol pipe; The machine stops, the cause of failure is discovered and corrected, and then it goes on again. The mechanism of an aeroplane may go wrong for similar reasons, but in this case the result is usually permanent, and usually the same–a mass of crumpled aluminium. We do not, however try to prove a common cause, the causes are as varied as those that stop an automobile but a secondary factor, that of gravity comes in to make what is temporary break-down in the first case, a permanent disaster in the second" (Ogilvie).

It is probable, then, that the causes of ulcer are as many and varied, but while lesions on the skin and mucous membranes are allowed to heal, those on a surface exposed to acids are digested away and become chronic ulcers, similar in each case whatever their first cause in ulcer production may be.

The only other recent advance regarding aetiology is the relation between blood groups and peptic ulcer. Although the evidence is inconclusive and disputed by Averyjones, it has been suggested, that blood group O individual more usually develop duodenal ulcer than one belonging to other blood groups. Averyjones gives an admirable succinct account of the correlation between the ability to secrete A. B. O. blood group antigens in saliva and other body fluids and the incidence of duodenal ulcer. In has been suggested that blood group antigens may confer some protection against duodenal ulceration, in as much as the incidence of duodenal ulcer is significantly higher in those who are unable to secrete them in body fluids "Secreting type" and "Non secreting type."

Tumours of alfa cell produce ulcers—alfa cells secrete an hyperchlorhydric hormone—Insulin hypersecretion may be due to this hormone in addition to the vagal action — (Experiments by Dr. V. Srinivasan Ph. D.)

The fallacy of fractional test meal analysis is too well known and it has almost been given up as a method of investigation in various clinics. There is no specific pattern of secretory response to a test meal which can be regarded as normal. The free acid level in health may range from O to nearly 100 clinical units. But a method has been devised in Edinburgh which has eliminated all possibilities of error. It is called histamine test. Patient fasts from 10 P. M. from the previous night and the Levine tube is passed and the patient screened to confirm the presence of the tube in the stomach before 9 A. M.

Histamine ·04 mgm per kilo gram is given intramuscularly and continuous suction of the stomach by a suction pump is done for 1 hour, and the total acid is calculated in M. eq. per litre. If it is more than 45 m. eq.—they say the patient will not respond to medical treatment and must undergo only surgical treatment straightaway. There were cases demonstrated with 80 to 90 m. eq. of H.C.L. who developed stomal ulcer even after repeated gastrectomies and ultimately total gastrectomy had to be done. The dosage of histamine is supposed to stimulate all the oxyntic cells to their maximum capacity which gives us an idea of maximum acid that a patient is capable of secreting. The theory is propounded that every individual is born with a specific number of oxyntic cells which do not increase. So we are almost born with an ulcer if the cells are too many! The results of these investigations are still not published They are confident of predicting not only failure of medical treatment but also specify the type of surgical treatment, and also the amount of stomach to be removed if gastrectomy has to be done. They are even counting the cells in the gastrectomy specimens, and calculate the total secreting cell mass left behind.

Diagnosis:- Regarding the diagnosis we have to diagnose all cases on clinical

grounds alone as it is quite possible to diagnose every case by careful and patient history taking which is most important. The pain of peptic ulcer will occur in bouts, which rarely lasts less than a month, seperated by intervals of freedom lasting anything from a month to several years.

During the bout, the pain usually recours regularly day after day with no holidays during the intervals it leaves no reminder. Gastric pain which disappears in a day or two, is rarely due to an organic lesion of the stomach or duodenum. Long remissions favour duodenal, and short ones gastric ulcers. A clock like punctuality is suggestive of gastric or duodenal ulcer. Unfortunately in our country as far as I know even in teaching hospitals the radiological diagnosis of peptic ulcer is to say the least, very unsatisfactory. It is a comon experience of every surgeon to find huge gastric ulcers on the table, which is not seen in the Barium meal skiagram. It is by mere accident that in some cases the ulcer is in a such a situation that it fills and is projected in the two dimensional picture. In foreign countries more than 99 per cent of both gastric and duodenal ulcers are demonstrated with the direct evidence of ulcer crater or niche and followed up to assess healing by medical treatment. The reason for this is obvious. It is not due to lack of knowledge, equipment or efficiency but to lack of effort.

Management :- As regards the management of peptic ulceration which is disabling but not necessarily fatal, it is our duty to advise that treatment which is most safe, simple and satisfactory. Medical treatment which has or rather which admits no mortality is therefore the first choice. It is still the first choice, but of late there has been a notable swing of opinion towards surgery, a tendency to resort to it more often and earlier. The reason for this change is two fold (1) That medical treatment though simple and safe has not proved particularly satisfactory and that (2) Surgical treatment though it is less simple than formerly, is very much safer, more satisfactory than it used to be.

The complete elimination of hydrochloric acid would abolish peptic ulcer, regardless of tissue susceptibility and other possible aetiological factors. However no method, pharmacological medical or surgical except total gastrectomy, consistently produces complete and permanent anacidity. Hence no procedure per se regularly cures the disease. Whatever may be the medical regime, it must be thorough, prolonged systematic, supervised and subject to discipline if it is to succeed.

Medical treatment : Carbonic anhydrase antagoniser acetazoliamide (Diamox) is tried to reduce the acid secretion but it does not act by oral route and so useless. Continuous sleep for 20 hours out of 24 hours in a day for 2 to 3 weeks was tried by Russian workers. Enterogastrone depresses the secretion of acid. Anticholinegic drugs should not be used when there is pyloric obstruction cariac ischamia prostatic obstruction and glaucoma-cortisone, phenylbutazone, rawolfia serpentina group of drugs and asprin will reactivate ulcer. Relief of pain does not correspond with healing of ulcer. The first essential is rest – the avoidence of mental strain and physical fatigue. There must be abstinence from many of the few remaining pleasures of our humdrum lives in the shape of good food, good wine, and nicotine." And the medical regime usually entails the ingestion of drugs and medicines – the antacids, the antispasmodics and vitamins. It is a prolonged, monotonous and costly discipline which is beyond the scope of many of our patients due to purely economic reasons – unlike in foreign countries where they are not prepared to forego the so called pleasures of life. Is there any

wonder then, that given time 85 percent of patients so treated will relapse ? Not, surely because that methods are not good but simply because they are not practical politics in our conditions. There is not enough money, not enough time to cope medically and adequately with the problem as it is to day. This naturally presumes: (1) That surgery is both relatively and reasonably safe and (2) That it produces the required results. If these two postulates can be satisfied, then we would press for the drastic curtailment of that so often long drawn out period of almost permanent medical treatment which today is almost impossible to institute properly for majority of our patients, and during which the patient is transformed from a relative, hopeful dyspeptic, to an equally hopeless wreckly a burden to himself and those around him, an economic wastage.

This swing towards surgical therapy has accelerated during the last 10 to 15 years and it has received more support from physicians than from Surgeons. This is undoubtedly so in the case of gastric ulcer–the more dangerous and fatal of the two. This tendency is independent of the much debated possibility of metamorphosis from gastric ulcer to gastric carcinoma. In respect of the gastric ulcer there is consensus of opinion that if it is seen denovo, it should be given an initial chance of months rather than of years to respond quickly and adequately to a good medical regime. If this trial fails completely or in part, surgery should be advocated and this should apply forthwith to the ulcer seen for the first time, with a long recurrent history (which we usually find) – particularly if its position of the stomach is on the greater curvature or more distal lesser curvature. We can do this with out hesitation because we know that patients will live happily ever afterwards. The cause of origin of gastric ulcer may be unknown but the cause of persistance is in the stomach itself. The failures of gastric surgery are all after operations for duodenal ulcer.

No doubt in duodenal ulcer the cause of origin and of the persistance of symptoms are in the patient – in his psychological make up, his job, and his habits. If a duodenal ulcer is as you believe is due to a turmoil that affects alike the cerebrum, the mid brain the autonomic system and the harmones which they control, the treatment is primarily that of patient and only incidently that of lesion. So the physician the psychiatrist, the wife, the business associate, the trusted friend, all can be allowed to have a chance before a surgeon comes in to the picture. Unless cure results by the philosophy that comes with advancing years the medical management of duodenal ulcer also is most often a failure in a great majority of our patients, purely due to economic reasons. This would so far seem high power surgical propaganda but I do this only because of the impracticability of the medical regime.

Admitted that the duodenal ulcer is not such fatal disease, that the bogey of carcinoma is absent, and that probably. at all events in the earlier stage the periods of natural remissions of symptoms are longer, even so, most duodenal, ulcers eventually follow a similar pathological course to that of gastric ulcers, including, one feature not common to gastric ulcer – obstruction. A young patient with duodenal ulcer – with a relatively short history and mild symptoms in affluent circumstances (particularly if the ulcer is uncomplicated) is fit for medical treatment ; but I can see no reason for varying the course previously mentioned for gastric ulcer, should the response be not both definite and rapid. To let a duodenal ulcer, simply because it is not likely to be fatal, become intractable and complicated is to let it become intolerable to the patient and a reflection on his medical attendants. It is a question of being forewarned and forearmed.

Evolution of Surgical Treatment :

— Gastro Jejunostomy
— Gastrectomy (Balfour, Moynihan. Mayo and Finisterer are pioneers)

— Vagotomy
— Vagotomy and Gastro Jejunostomy
— Vagotomy and antrectomy
— Colonic transplantation
— Vaso ligation (Wilson Hay and Somervile) and Gastrojejunostomy.

Surgical treatment. Having argued the case for surgical treatment in the majority of cases, it is our duty to make it sure, safe, simple, and satisfactory.

Under conditions obtained in the west, where both the Surgeon and the patient are suited for the radical procedure of partial gastrectomy which probably is the treatment of choice for reduction of acid, I feel that a less dangerous, and less mutilating operation which gives equally satisfactory result vix vagal resection and gastroenterostomy is suited for our conditions.

When vagectomy first appeared on the surgical horizon, it seemed the herald of a new era in the surgical treatment of ulcer; but soon became obscured by distrust from which it is only now emerging. As has happened to so many other valuable surgical advances, the operation fell into disrepute through over enthusiasm for a new method of treatment and lack of technical experience. It was definitely found that bad results of vagectomies are due to bad vagectomies i. e. incomplete nerve sections.

A great deal of re-search both clinical and experimental has been carried out on the results of vagotomy since Dragstedt performed the first really successful operations on human subjects in 1943. The recorded vagotomies in the world literature now total many thousands and sufficient accurate information is now available.

The approach to the problem must be strictly logical; though essentially critical, it must be unprejudiced by any individual bias. Neither personal preference for an alternative operation, nor life time experience in the technique of any particular procedure should be allowed to cloud the issue.

Gastrectomy being a more serious and mutilating operation did not and does not supply the answer, especially for our patients. Extirpation of a useful organ is accepting defeat. Here the reduced acidity is obtained at a great cost of reduced stomach capacity and digestive capability. Lester. R. Dragstedt of Chicago after an experience of more than nine years during which he has done 800 vagal resections is of opinion that this is a safe, efficient and practical method of surgical treatment and should replace subtotal gastrectomy as the initial definitive surgical treatment for duodenal-gastro jejunal and certain oesophagual ulcers. It should be combined with posterior gastro jejunostomy of small size i. e. 1·5 cms. wide-6 cms from pylonus to facilitate emptying of the stomach.

The therapeutic value of this operation depends upon the fact that it abolishes.

1. Nervous phase of gastric secretion and also chemical.

2. Reduce markedly the total output of gastric juice.

3. Reduce the tonus and motility of the stomach without causing serious complications or side effects and without loss of storage function of the stomach. Complete interruption of all the vagal fibres is necessary.

Recent work of Dragestedt has shown that the duodenal ulcer is prevented from healing by the vagal cephalic phase of secretion while gastric ulcer is prevented from healing by the normal phase.

Crile of Cleveland Clinic has reported a large series of cases with encouraging results.

The indications are almost same as for partial gastrectomy for duodenal ulcers.

Advantages as compared with partial gastrectomy especially in the labouring class of people. in our country, who are used to large bulky food and cannot afford frequent nutrious diet, are :

1. Mortality: In expert hands 1 to 2. per cent ; must be much more in our country. Vagectomy carries almost no mortality or post operative morbidity.

2. Recurrence Rate: The proved recurrence rate after gastrectomy varies from 2 to 6 per cent. In this series of vagectomy and G. J. the recurrence rate was 4–25 per cent. This includes incomplete resection.

3. Nutritional state: Loss of weight quite common in gastrectomy. Post Operative anaemia: Quite common after gastrectomy. Most of our patients neglect it due to economic reasons and it causes considerable disability.

4. Physical state : Much better after vagectomy. Not many patients are fit for heavy manual work after gastrectomy.

5 **Change of Bowel Habit:** 30 per cent have diarrhoea after vagectomy and 20 per cent even after gastrectomy but not troublesome.

6. **Post Gastrectomy Syndrome:** The symptoms and in some cases great disability after gastrectomy is too well known – Much less after vagotomy. The late hypoglycaemic episodes are said to be due to disturbance of carbohydrate metabolism due to denervation of liver and pancreas in both operations but the lesser incidence after vagectomy is unex plained.

So it is concluded that vagectomy with gastrofureslong is a better operation and the operation of choice in either sex and at any age when surgery is indicated for chronic duodenal ulcer, especially in young and also in women who respond badly to gastrectomy as the demand on their haemopoietic systems is great due to pregnancy menstruation etc.

7. Another advantage claimed now is immunity from reflex digestive instability that accompanies nervous stress. The possibility of doing it in any moffussil hospital without the necessity of blood transfusion etc. must also be considered.

The liteature dealing with aetiology and treatment of peptic ulcer is not only overwhelming, but is self cancelling. Whatever we wish to believe, we can find evidence in experimental work to prove theory. So we have to choose treatment to suit our conditions.

POST TRAUMATIC KNEE JOINT DISEASES

by

Prof. Dr. SOPHUS VOM ROSEN,
Professor of Orthopaedics, University of London and Surgeon in Chief, Orthopaedic Department, Malmo General Hospital, Sweden.

THE subject mater contains nothing new in itself. It is intended as a guide to the manner in which orthopaedic specialists in the Scandinavian countries regard these diseases and it is a summary of my personal experiences after more than 25 years work in orthopaedics.

From the orthopaedical point of view, the knee joint is a particularly important joint. It is often exposed to trauma of different types not least during play and sport (handball and football and skiing). Injuries which occur during skiing are certainly not always sport injuries for in the northern part of Sweden skiing is a natural form of transport for the people during the winter months.

Post traumatic knee joint diseases are relatively uncommon in children.

In the diagnosis of a knee joint injury the type of trauma may be decisive. The knee joint responds to a trauma by a discharge which may be a hydrops or a bleeding in the joint (hemarthrosis). From the diagnostical and also prognostical stand-point it is important to know which type is present.

A post traumatic hydrops occurs gradually and is often not noticed by the patient until the day after the accident. Hemarthrosis often gives trouble a few hours after the trauma.

The examination of a knee joint comprises first inspection. Is it driven upward? Are the patellar contours normal? Is there deformation of the knee? (Valgus position? Varus position? Flexion? Subluxation?) Is muscle atrophy of the thigh present?

Palpation: Is there increased warmth? Tenderness under pressure? Shifting of the patella? This last simply means that a discharge is present in the joint.

Mobility: How is the active mobility? This generally remains within the limits applied by painful movement. How is the passive mobility within the normal range of movement? Is there pathological mobility.

When examining a knee joint it should always be compared with the knee joint of the other side.

In Scandinavia we include roentgen examination, sometimes in the form of arthrography, very often as a supplimentary examination.

If a discharge is present a test puncture of the joint may give valuable information An absolute requisite is that one must be able to rely on its sterility. If from a test puncture infection of the joint should occur, the situation is fatal even if in these times such a position can be controlled better than before, thanks to antibiotics. If a puncture reveals blood in the joint one may conclude that the injury is of a more serious nature.

A simple traumatic hydrops, usually caused by wrenching of the knee joint

does not require any special treatment. It recedes after about a week with or without elastic or half supporting bandage round the knee.

Behind an apparently simple post traumatic hydrops there *may* exist a meniscus or ligamental injury.

Meniscus injuries.

Most common is the injury to the medical meniscus. The typical trauma is that the lower leg has been pushed outward (in valgus direction) and simultaneously rotated outward in relation to the thigh bone. The meniscus can become loose forward and backward or suffer a longitudinal rupture at or just inside the attachement of the capsule. The part released may be pressed into the joint and lock it. If the meniscus is loose inside the capsular attachement bleeding may occur in the joint. If cracks are formed in the meniscus cartilage itself no bleeding occurs in the joint but a hydrops may appear.

A loose meniscus does not generally become firmly attached again. It leads, one may surely say sooner or later, to locking of the joint which once begun usually recurs at shorter and shorter intervals. When a loose meniscus is dislocated into the joint this is shown by an extension defect in the joint of perhaps 10—15°.

A dislocated meniscus can sometimes be reduced by the patient himself through various manipulations, sometimes with the aid of another person who pulls and works at the lower leg. When the meniscus slips into place again this can be heard of the patient feels a click and move the knee freely again.

The surest signs of the existence of a meniscus injury with luxation tendencies are found in repeated locking attacks in the case history and, combined with locking, an extension defect in the knee joint. Pain on the inner side of the knee combined with locking and tenderness on pressure medially above the joint space also appear amongst the symptoms.

A normal roentgen examination does not give direct information concerning a meniscus injury but it is valuable insofar as other injuries apart from the actual conditions can be excluded e. g. intra-articular fracture, joint mice, etc.

Arthrography can produce direct information about a meniscus injury but unfortunately not too often in such cases where the clinical diagnosis is not already clear.

How is the meniscus injury to be treated ? If the diagnosis is clear the meniscus injury should be operated on and the injured meniscus be removed. Locking symptoms are often very troublesome and obstructive to the patient and the prognosis for operative intervention must be considered as good. In cases where operation does not produce freedom from trouble this is often due to the presence of other injuries beside the meniscus injury: these may possibly have been overlooked or also not been capable of repair. I am thinking especially about chondromalacia patellae or injuries to the medieal collateral ligaments of the knee or leg, cruciat anterior.

Injuries to the lateral meniscus are less common. The kind of trauma is less typical. In attacks of luxation the region of trouble is felt more laterally in the knee joint and the patient feels tenderness laterally above the joint space Injuries to the lateral meniscus sometimes occur even in children. The injury to the lateral meniscus ought to be operated on also, particularly if it has led to an extension defect in the knee joint. The prognosis for this operation is not quite as good, probably because the lateral

meniscus possesses greater importance for the congruence between the articular surfaces of the knee than the medial meniscus.

Ligamental injuries.

Injuries to the stabilizing ligaments of the knee are relatively common, sometimes as isolated injuries, sometimes combined with other injuries. If in an accident the lower leg is wrenched outward (in valgus direction) a rupture may occur of the knee's medial collateral ligaments. On examination an instability is found in the joint (in the frontal plane) and also tenderness above the ligament. The rupture may be total or partial.

Treatment consists in fixation of the knee joint in an extended position for 6-8 weeks or possibly in operation with suturing of the ligament. If the injury is recent, suturing of the ligament is a simple matter although not always ne cessary. The conservative, nonoperative treatment is based on experience that scar tissue arises at the rupture site which restores the stability of the knee.

Injuries to the lig. cruciata.

As is well-known the lig. cruciata lie inside the knee joint. They give the joint stability in the sagittal plane. Most common is injury to the anterior cruciata ligament. The rupture is often sited on the ligament's attachment to the anterior intercondyloid eminence. The ligamental attachment is pulled out like a cork from a bottle. In such cases bleeding occurs in the joint, a hemarthrosis, in other cases, a traumatic hydrops. If the anterior cruciate ligament is torn away the knee joint becomes unstable.

Examination is made with the knee flexed about 90°. If the ligament is torn away the lower leg can be pulled forward perhaps a few centimetres in relation to the famur. The lower leg can be drawn forward into a subluxation position. Such an instability may appear very plainly on examination, in other cases it is doubtful. The other knee should always be compared to make certain that a clear difference exists between both sides. In the diagnosis the X-rays may be decisive in those cases where the injury is combined with a rupture of the ligament attachment. The dragged-up and dislocated intercondyloid eminence emerges clearly on the X-ray. Sometimes an arthrography may show rupture of the ligament.

How shall a diagnosed injury to the anterior cruc.lig. be treated? In answering this question one must consider the point that injuries to the anterior cruc. lig. often do not give any trouble at all to the patient. On the other hand an injury to the anterior crug. lig. gives a number of patients an uncomfortable feeling of uncertainty in the knee. In my opinion these injuries should be operated on, therefore if the diagnosis is completely clear. However this should be done during the first weeks after the injury is sustained; Later on the ligament stumps are retracted so that suturing becomes impossible. In practice it is almost solely those injuries sited on the anterior attachment of the ligament, giving a definitely positive roentgen picture, which are accessible for operation. At operation the ligament attachment is drawn down into place and fixed there in a suitable way. After such an intervention full mobility and full stability of the knee can be anticipated. If a large part of the tibial condyle has been dragged loose this fragment can sometimes be reduced and kept in position during healing by fixing the knee in extreme extension. This also applies to children who now and again manifest such injuries.

If an old injury to the anterior cruc. lig. has led to troublesome instability for the patient a ligamental plastics may restore stability, generally however, at

the cost of the knee's mobility. In such cases a range of movement of about 90° may yet be obtained.

Rupture of the posterior cruc. lig. is more uncommon. It leads to a subluxation backwards of the tibia in relation to the femur. On examination of the symptom the tibia can be pulled forward into the normal mid-position. Operation on such an injury is seldom performed.

Tibial condyle fractures.

Fracture of the tibial condyle is common and important in practice. The trauma is often of the same type as in meniscus injuries. Three different types of fracture can be distinguished.

1) A fracture of the lateral tibial condyle with pressure outward of the detached lateral part of the tibial condyle.

2) A compression fracture of the lateral tibial condyle with downward pressure of greater or lesser parts of the condyle.

3) A combination of the fracture types described above or a T-shaped fracture. The lateral meniscus is often torn loose and lies interposed in the gab of the fracture.

The tibial condyle fracture produces hemarthrosis with drops of fat from the bone marrow entering the blood. For diagnosis one relies on the roentgen. examination.

How shall the tibial condyle fracture be treated? The main principle is the same as in other intra-articular fractures in that it is important to restore the articular surfaces as well as possible to avoid permanent incongruence of these surfaces. Such incongruence leads sooner or later to deforming arthrosis, as far as we know because the weight borne by the joint cartilage is not uniform. The weight bearing joint cartilage is worn down more quickly than that which is not exposed to the same strain.

In the clinic in Malmo these fractures are operated on if no success has been obtained in reducing the fracture by pulling and pressing from outside. This succeeds sometimes but not too often. Generally on operation a good reduction position can be obtained. Unfortunately however one sees now and again that the tibial condyle becomes deformed after some years owing to aseptic necrosis and resorption of the injured part of the condyle.

If a tibial condyle promotes a valgu position, of the knee, or sometimes a varus position, a corrective osteotomy may restore the normal shape of the leg.

Injuries to the extension mechanism of the knee.

These injuries are ruptures to the quadriceps tendon, fractures of the patella and ruptures of the patellar ligament. Amongst these injuries the patellar fractures are the most common.

An injury to the extension mechaism of the knee with interruption of its continuity produces an incapacity to lift the leg with a straight knee. At the point affected by the injury an irregular depression occurs owing to a diastasis between the injured tissues and also a distinct tenderness to pressure at this site.

If the patella is fractured the X-rays give information about the appearance of the injury. A fracture of the patella may also arise without interruption of the knee's extension ability. This is especially so if the fracture is sited on the tip of the patella, i.e. the part of the pattella which has no direct connection with the

joint space. A patella fracture usually leads to bleeding in the joint, a hemarthrosis.

The object of treatment is in principle to restore the extension ability of the knee. The tendon rupture must be sutured as quickly as possible after the occurrence of the injury. This is because a diastasis arises very soon at the site of rupture owing to shrinking of the tendon ends and contraction of the quadriceps musculature.

If interruption in continuity is the result of a patella fracture there are various methods of treatment. It is then important not only to restore the extension ability of the knee but also to obtain an even cartilage surface on the posterior aspect of the patella. This can rarely be achieved without operation.

An osteosynthesis of the patella can be performed in various ways. If when operating on a patellar fracture a level cartilage surface cannot be achieved the patella ought to be completely exstirpated. It has been feared that this would lead to deforming arthrosis in the future. These fears are according to my experience, unjustified. I have had the opportunity of examining patients whose patellas were exstirpated more than 20 years ago and who have complete function of the knee joint and no sign of deforming arthrosis. The risk of a deforming arthrosis occurring after a patellar fracture is quite certainly much greater if a patella deformed by fracture and with a rough uneven surface is allowed to remain in the joint. Moreover the after-care is much simpler if the patella is removed; good mobility in the knee joint is much more quickly achieved. The practical consequence of my experience is, therefore, that the patella ought to be removed in its entirety in comminuted patellar fractures and also in patellar fractures suffered by older people, let us say, those over 50 years of age.

CASE NOTES

Cases of Abdominal Emergencies.

During the period of four months 28 cases of abdominal emergencies (excluding those of an obstetric or gynaceological nature, but including strangulated hernia) were treated by operative measures in this hospital. They are classified as follows :—

		(For Four Months)
Strangulated Hernia	..	10 cases
Perforated Peptic Ulcer	..	7 ,,
Acute Appendicitis	..	3 ,,
Abdominal Injuries	..	6 ,,
Burst Abdomen	..	1 ,,
Mesenteric Thrombosis	..	1 ,,

Of the cases of *Strangulated Hernia* in the above series, five presented factors of unusual interest. One was an obstructed inguinal Hernia in a fatty middleaged female. The Hernia had descended into the labium majus and had become obstructed at the external ring but there was no evidence of Strangulation. The viable contents were reduced after release of the obstruction and a herniorrhaphy done in this case In another case of extensive gangrene following strangulation at the neck of the hernial sac a *hemicolectomy* had to be done followed by the necessary ileo-transvers colostomy. In one case, the damage was not so much and resection of one foot of the ilium was carried out. In yet another case of strangulated hernia on opening the sac, purulent fluid was found in the sac and the contents were found to be a matted loop of small intestine with a perforation at its apex plugged completely by omentum. In this case, a diagnosis of perforation of the small intestine inside the hernial sac followed by localised peritonitis was obvious. In one case, the strangulation was observed in a sliding hernia. Usually the neck of a sliding hernia being very wide, obstruction or strangulation in such a case is a rare phenomenon. Of the seven cases of perforated peptic ulcers, five were duodenal and two gastric.

Among the cases of acute appendicitis, one was associated with pregnancy. Another case, also in a female patient, presented remarkable features on operation. On opening the abdomen through the obilique Rutherford-Morrison incison usually employed in this hospital, the caecum was found to be adherent to the surrounding structures to some extent and the appendix was missing at its site. In its place the caecum showed a small perforation. Under the assumption that the appendix had perforated at its base and sloughed off, a search was made for the appendix in the region, but proved futile. On passing a probe through the perforation, it was found to lead not into the caecal cavity, but into a cul-de-sac situated on the anteromedial wall of the caecum. Evidently, the appendix in this case was paracaecal and had become burried by adhesions in the caecal wall after inflammation. Appendicectomy was done after isolating the organ by dissecting into adhesions over the caecal wall.

Among the 6 cases of abdominal injuries 2 were due to bullgore, 2 due to sharp instruments and 2 due to heavy vehicular accidents. In the cases of bullgore there were no visceral injuries. In one case of injury by sharp instrument, the patient a working girl had fallen from a bench on to a sharp-pointed scissors in a beedy factory and sustained an injury in the subumbilical region. On extending the wound and exploration, a large collec-

Reprinted from the Proceedings of the North Arcot District Clinical Society 1957,

tion of blood was found in the peritoneal cavity, but there was no visceral injury. Further exploration revealed that the site of bleeding was an omental vessel. The injured omenttum was excised and the incision closed.

Of the remaining two cases, one was an old man who had sustained a fall from a bullock cart and who showed a perforation of the jejunum on laparotomy. The other case was a small ten year old boy who was run over by a car. On laparotomy in this case also there was a rupture of the upper loop of jejunum.

The solitary case of burst abdomen occurred in an epileptic patient who had violent convulsions on the 12th day after a gastro-jejunostomy was done. Fortunately timely closure saved him and he had an uneventful convalescence.

The rare case of mesenteric vascular occlusion was diagnosed only after laparotomy. The patient a well built man of 35 years walked into the out-patient department with a complaint of dull abdominal pain of 8 hours' duration without any vomiting, constipation abdominal distension or rigidity. He was admitted for observation and within 12 hours he developed abdominal distension, rigidity of the whole lower abdomen, a temperature of 100° and a very rapid pulse. Rectat examination did not reveal anything significant. Laparotomy was carried out immediately and on opening the abdomen more than 12 feet of small intestine was found to be gangrenous and the omentum destroyed into shreds. There was no evidence of any obstruction or volvulus and a diagnosis of mesenteric vascular occlusion was made.

An attempt has been made here to describe in some detail a few of the abdominal emergencies which presented peculiar and interesting features and proved the truth of the saying that the "abdomen is a temple of surprise".

Treatment of Diabetic Coma

Prevention of diabetic coma is much more important than treatment itself as it will prevent all complications and thus reduce mortality. Diabetic coma a few years ago meant death. But thanks to our present knowledge of metabolism, we are able to save most of our cases. Thus, in prevention, a systamatic examination by the doctor when the patient attends for his regular injections is of prime importance. At every attendance of the patient, the doctor should go through the following:

(a) Symptoms if any.

(b) Changes in weight.

(c) Signs of de-hydration

(d) any sugar or ketone bodies in a specimen of urine collected first thing in the morning. Some times, it is better to examine a 24-hour specimen of urine.

But, if these precautions had not been taken and the patient has reported in a precomatose state or in coma with unconsciousness, we should proceed in the following manner:

Diabetic coma is a medical emergency, requires prompt treatment and that energetic. A thorough understanding of the physiological and pathological processes will help in the treatment. Thus, we should overcome with all speed (a) Ketosis by means of Insulin, (b) De hydration and circulatory collapse and sodium depletion by means of fluids, (Saline) (c) Vomiting by means of gastric lavage and (d) infection by means of anti-biotics. Here, it will not be out of place to say that most of the comatose condition in diabetics are due to some inter current infection which raises the requirements of insulin for the patients and which has gone unnoticed.

In coma and dehydration, the patient is resistant Insulin and noting this, we should give the high doses of insulin. Further, the tissues are in such a state that they cannot absorb parenteral insulin and as such, a good quantity of the calculated dose must be given by the intravenous route. Immediately, on seeing the patient and one having diagnosed the case as diabetic coma, 50 units of insulin should be given by the intravenous route. Here, for a diagnosis of Diabetic Coma, a catheterised specimen of urine should be used. An average case may need anywhere from 250 units or more in the first 24 hours and few cases require nearly 1,000 units. The mortality is accordingly reduced by giving insulin intravenously. In such cases, at least one third of the calculated dose should be given by this route. Saline should be given in plenty and about 2 litres should be introduced as early as possible. Even if the patient is able to swallow fluids, saline is better given by the intravenous route because the stomach in these conditions is rather lethargic and may not pass on the fluid for absorption lower down and even in the intestines, absorption may be very sluggish. The treatment must be controlled by repeated tests for sugar in urine and acetone and estimation of blood sugar every 24 hours. The carbon dioxide combining power of the blood is of great value to indicate acidotic condition of the blood. Here, a digression is permissible that estimation of blood sugar is not a necessity for diagnosis of diabetes as such except in very very rare conditions whereas in treatment of coma, blood sugar is of prime importance.

Vomiting is troublesome in this condition and coma may be precipitated by gastro intestinal upsets. Thus, washing out the stomach with weak saline or sodabicarb is of prime importance not only to stop vomiting but also to replace some amount of sodium lost by vomit.

As coma is precipitated nearly always by infection somewhere in the body, a control of infection by penicillin is necessary. High doses of penicillin should be given to these cases. As soon as consciousness returns, the patient should receive by mouth fluids containing 25 to 40 grams of carbohydrate and enough insulin to cover the same.

There is also a great danger of: Hypopotassemia occurring in the treatment of diabetic coma. When insulin in large doses is given, it carries the potassium away from the plasma into the tissues and a relative hypo-potassemia results. Repeated vomits lose a lot of potassium also. Here, giving of intravenous potassium is not good or perhaps is dangerous as it may result in Hyper-potassemia. Thus, when the gastric lavage is done, it is much better to leave potassium salts in the stomach. Later, when the patient has become conscious, potassium citrate 2 grams should be given every four hours. Without the above treatment the patient may be in coma indefinitely and ultimately die of Ketosis or from circulatory failure or from renal failure resulting in anuria.